

## Freeform Search

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<b>Database:</b>	US Pre-Grant Publication Full-Text Database US Patents Full-Text Database US OCR Full-Text Database EPO Abstracts Database JPO Abstracts Database Derwent World Patents Index IBM Technical Disclosure Bulletins
<b>Term:</b>	l6 and (mu near5 (nucleic acid\$1 or DNA\$1))
<b>Display:</b>	10 Documents in Display Format: - Starting with Number 1
<b>Generate:</b> <input type="radio"/> Hit List <input checked="" type="radio"/> Hit Count <input type="radio"/> Side by Side <input type="radio"/> Image	

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### Search History

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**DATE:** Tuesday, March 06, 2007   
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<u>Set</u> <u>Name</u> side by side	<u>Query</u>	<u>Hit</u> <u>Count</u>	<u>Set</u> <u>Name</u> result set
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>			
<u>L8</u>	l6 and (mu near5 (nucleic acid\$1 or DNA\$1))	14	<u>L8</u>
<u>L7</u>	L6 and (mu near5 nucleic acid or DNA)	138	<u>L7</u>
<u>L6</u>	L4 and (site\$1 near5 (muta\$4 or varian\$3 or delet\$3 or insert\$3 or mismatch))	139	<u>L6</u>
<u>L5</u>	L4 and site\$1 near5 (muta\$4 or varian\$3 or delet\$3 or insert\$3 or mismatch)	139	<u>L5</u>
<u>L4</u>	(detect\$3 or determin\$3) near5 transposit\$3	560	<u>L4</u>
<u>L3</u>	L2 and (mu or mutator)	10	<u>L3</u>
<u>L2</u>	transpos\$5 near5 detect\$3 (mismatch\$3 or delet\$3 or insert\$3 or varian\$3)	18	<u>L2</u>
<u>L1</u>	+	968764	<u>L1</u>

END OF SEARCH HISTORY

STN search

AN 2004:803873 CAPLUS

DN 141:290033

TI Methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis

IN Yanagihara, Katsuhiko; Mizuuchi, Kiyoshi

PA United States Dept. of Health and Human Services, USA

SO U.S. Pat. Appl. Publ., 24 pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004191821	A1	20040930	US 2004-809688	20040326
PRAI	US 2003-457934P	P	20030328		

AB Phage Mu DNA transposes to duplex target DNA sites with limited sequence specificity. The present invention provides methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microbes using mismatch-targeted Mu transposition for use in diagnosis.

Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This finding has implications for the mechanism of transposition and provides a powerful tool for genomic research. A single mismatch could be detected as a preferred target of Mu transposition in the presence of 300,000-fold excess of non-mismatched sites. Both heterozygous and homozygous mutations in the cystic fibrosis transmembrane conductance regulator gene and single nucleotide polymorphism in HLA region were detected by Mu transposition mismatch anal. procedure.

TI Methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis

AB Phage Mu DNA transposes to duplex target DNA sites with limited sequence specificity. The present invention provides methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microbes using mismatch-targeted Mu transposition for use in diagnosis.

Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This finding has implications for the mechanism of transposition and provides a powerful tool for genomic research. A single mismatch could be detected as a preferred target of Mu transposition in the presence of 300,000-fold excess of non-mismatched sites. Both heterozygous and homozygous mutations in the cystic fibrosis transmembrane conductance regulator gene and single nucleotide polymorphism in HLA region were detected by Mu transposition mismatch anal. procedure.

ST mismatch targeted transposition phage Mu detection  
polymorphism mutation; gene HLA CFTR phage Mu transposition  
cancer diagnosis pathogen

IT Gene, animal

RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(APC, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal

RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(BRCA1, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal  
 RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (CFTR, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal  
 RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (HLA, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal  
 RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (HMLH1, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal  
 RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (HMSH1, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal  
 RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (RBL, mutation in; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gene, animal  
 RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (TP53; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gel electrophoresis  
 (acrylamide or agarose; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Diagnosis  
 (cancer; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Gel electrophoresis  
 (capillary; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Pathogen  
 (detection of; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Capillary electrophoresis  
 (gel; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Enterobacteria phage Mu

High throughput screening

Human

Mutation

Neoplasm

PCR (polymerase chain reaction)

Susceptibility (genetic)

Tumor markers

(methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Primers (nucleic acid)

RL: ARG (Analytical reagent use); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Diagnosis

(mol.; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Genetic polymorphism

(single nucleotide; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Enzymes, biological studies

RL: ARG (Analytical reagent use); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(transposases, My; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT Recombination, genetic

(transposition; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT 79-06-1, Acrylamide, biological studies 9012-36-6, Agarose

RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)

(gel electrophoresis; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

IT 763155-35-7 763155-36-8 763155-37-9 763155-38-0 763155-39-1

763155-40-4 763155-41-5 763155-42-6

RL: PRP (Properties)

(unclaimed nucleotide sequence; methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microorganisms using mismatch-targeted Mu transposition for use in diagnosis)

L8 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2004:802450 CAPLUS

DN 141:290022

TI Target-dependent transcription using deletion mutants of coliphage N4 RNA polymerase (mini-vRNAP) and N4 promoter

IN Davydova, Elena K.; Rothman-Denes, Lucia B.; Dahl, Gary A.; Gerdes, Svetlana Y.; Jendrisak, Jerome J.

PA USA

SO U.S. Pat. Appl. Publ., 147 pp., Cont.-in-part of U.S. Ser. No. 153,219.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 6

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004191812	A1	20040930	US 2003-743975	20031223
	US 2003096349	A1	20030522	US 2002-153219	20020522
PRAI	US 2001-292845P	P	20010522		
	US 2002-153219	A2	20020522		
	US 2002-436062P	P	20021223		

AB The present invention comprises novel methods, compns. and kits that use coliphage N4 virion RNA polymerase (vRNAP) deletion mutants to detect and quantify analytes comprising one or multiple target nucleic acid sequences, including target sequences that differ by as little as one nucleotide or non-nucleic acid analytes, by detecting a target sequence tag that is joined to an analyte-binding substance. The method consists of an annealing process, a DNA ligation process, an optional DNA polymerase extension process, a transcription process, and, optionally, a detection process. The invention further claims DNA and protein sequences for N4 vRNAP and sequences for promoters. In examples of the invention, a transcriptionally active fragment of the N4 vRNAP, mini-vRNAP, was further characterized. VRNAP-promoter recognition and activity required specific sequences and a hairpin structure on the template strand. A reporter plasmid containing the N4 promoter P2 and lacZ' gene and a plasmid expressing mini-vRNAP under pBAD control were used to transform Escherichia coli DH5 strain. The invention has broad applicability for research, diagnostic and therapeutic applications, such as preparing cDNA corresponding to full-length mRNA, making sense or anti-sense probes, detecting gene- or organism-specific sequences, cloning, cell signaling or making RNA for use in RNAi.

AB The present invention comprises novel methods, compns. and kits that use coliphage N4 virion RNA polymerase (vRNAP) deletion mutants to detect and quantify analytes comprising one or multiple target nucleic acid sequences, including target sequences that differ by as little as one nucleotide or non-nucleic acid analytes, by detecting a target sequence tag that is joined to an analyte-binding substance. The method consists of an annealing process, a DNA ligation process, an optional DNA polymerase extension process, a transcription process, and, optionally, a detection process. The invention further claims DNA and protein sequences for N4 vRNAP and sequences for promoters. In examples of the invention, a transcriptionally active fragment of the N4 vRNAP, mini-vRNAP, was further characterized. VRNAP-promoter recognition and activity required specific sequences and a hairpin structure on the template strand. A reporter plasmid containing the N4 promoter P2 and lacZ' gene and a plasmid expressing mini-vRNAP under pBAD control were used to transform Escherichia coli DH5 strain. The invention has broad applicability for research, diagnostic and therapeutic applications, such as preparing cDNA corresponding to full-length mRNA, making sense or anti-sense probes, detecting gene- or organism-specific sequences, cloning, cell signaling or making RNA for use in RNAi.

IT Probes (nucleic acid)

RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)  
(for transcription detection; target-dependent  
transcription using deletion mutants of coliphage N4 RNA  
polymerase (mini-vRNAP) and N4 promoter)

IT Genetic element

RL: BUU (Biological use, unclassified); BIOL (Biological study); USES  
(Uses)  
(transposon recognition site; target-dependent transcription  
using deletion mutants of coliphage N4 RNA  
polymerase (mini-vRNAP) and N4 promoter)

IT Genetic element

RL: BUU (Biological use, unclassified); BIOL (Biological study); USES  
(Uses)

(transposon recognizing, ssDNA template containing;  
target-dependent transcription using deletion mutants  
of coliphage N4 RNA polymerase (mini-vRNAP) and N4 promoter)

L8 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2003:482975 CAPLUS

DN 139:144848

TI Target DNA Bending is an Important Specificity Determinant in Target Site Selection in Tn10 Transposition

AU Pribil, Patrick A.; Haniford, David B.

CS Department of Biochemistry, University of Western Ontario, London, ON, Can.

SO Journal of Molecular Biology (2003), 330(2), 247-259

CODEN: JMOBAK; ISSN: 0022-2836

PB Elsevier Science Ltd.

DT Journal

LA English

AB The bacterial transposon Tn10 inserts preferentially into specific DNA sequences. DNA footprinting and interference studies have revealed that the Tn10-encoded transposase protein contacts a large stretch of target DNA (.apprx.24 bp) and that the target DNA structure is deformed upon incorporation into the transpososome. Target DNA deformation might contribute significantly to target site selection and thus it is of interest to further define the nature of this deformation. Circular permutation anal. was used to demonstrate that the target DNA is bent upon its incorporation into the transpososome. Two lines of evidence are presented that target DNA bending is an important event in target site selection. First, we demonstrate a correlation between increased target site usage and an increased level of target DNA bending. Second, transposase mutants with relaxed target specificity are shown to cause increased target DNA bending relative to wild-type transposase. This latter observation provides new insight into how relaxed specificity may be achieved. We also show that Ca2+ facilitates target capture by stabilizing transposase interactions with sequences immediately flanking the insertion site. Ca2+ could, in theory, exert this effect by stabilizing bends in the target DNA.

RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB The bacterial transposon Tn10 inserts preferentially into specific DNA sequences. DNA footprinting and interference studies have revealed that the Tn10-encoded transposase protein contacts a large stretch of target DNA (.apprx.24 bp) and that the target DNA structure is deformed upon incorporation into the transpososome. Target DNA deformation might contribute significantly to target site selection and thus it is of interest to further define the nature of this deformation. Circular permutation anal. was used to demonstrate that the target DNA is bent upon its incorporation into the transpososome. Two lines of evidence are presented that target DNA bending is an important event in target site selection. First, we demonstrate a correlation between increased target site usage and an increased level of target DNA bending. Second, transposase mutants with relaxed target specificity are shown to cause increased target DNA bending relative to wild-type transposase. This latter observation provides new insight into how relaxed specificity may be achieved. We also show that Ca2+ facilitates target capture by stabilizing transposase interactions with sequences immediately flanking the insertion site. Ca2+ could, in theory, exert this effect by stabilizing bends in the target DNA.

ST Tn10 transposon transposase DNA bending calcium insertion site

IT Genetic element

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(insertion site of transposon; target DNA

Bending is an Important Specificity Determinant in Target

Site Selection in Tn10 Transposition)

L8 ANSWER 4 OF 11 MEDLINE on STN DUPLICATE 1  
 AN 2002432274 MEDLINE  
 DN PubMed ID: 12177413  
 TI Mismatch-targeted transposition of Mu: a new strategy  
 to map genetic polymorphism.  
 AU Yanagihara Katsuhiko; Mizuuchi Kiyoshi  
 CS Laboratory of Molecular Biology, National Institute of Diabetes, Digestive  
 and Kidney Diseases, National Institutes of Health, Bethesda, MD 20892,  
 USA.  
 SO Proceedings of the National Academy of Sciences of the United States of  
 America, (2002 Aug 20) Vol. 99, No. 17, pp. 11317-21. Electronic  
 Publication: 2002-08-12.  
 Journal code: 7505876. ISSN: 0027-8424.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 LA English  
 FS Priority Journals  
 EM 200209  
 ED Entered STN: 22 Aug 2002  
 Last Updated on STN: 5 Jan 2003  
 Entered Medline: 27 Sep 2002  
 AB Phage Mu DNA transposes to duplex target DNA sites with limited sequence  
 specificity. Here we demonstrate that Mu transposition exhibits a strong  
 target site preference for all single-nucleotide mismatches. This finding  
 has implications for the mechanism of transposition and provides a  
 powerful tool for genomic research. A single mismatch could be  
 detected as a preferred target of Mu  
 transposition in the presence of 300,000-fold excess of  
 nonmismatched sites. We demonstrate the detection of both heterozygous  
 and homozygous mutations in the cystic fibrosis transmembrane conductance  
 regulator gene and single nucleotide polymorphism in HLA region by Mu  
 transposition mismatch analysis procedure.  
 TI Mismatch-targeted transposition of Mu: a new strategy  
 to map genetic polymorphism.  
 AB . . . mismatches. This finding has implications for the mechanism of  
 transposition and provides a powerful tool for genomic research. A single  
 mismatch could be detected as a preferred target  
 of Mu transposition in the presence of 300,000-fold excess of  
 nonmismatched sites. We demonstrate the detection of both heterozygous  
 and homozygous mutations in the cystic fibrosis transmembrane conductance  
 regulator gene and single nucleotide polymorphism in HLA region by Mu  
 transposition mismatch analysis procedure.

L8 ANSWER 5 OF 11 MEDLINE on STN DUPLICATE 2  
 AN 1998315086 MEDLINE  
 DN PubMed ID: 9649512  
 TI UV light induces IS10 transposition in Escherichia coli.  
 AU Eichenbaum Z; Livneh Z  
 CS Department of Biological Chemistry, Faculty of Biochemistry, The Weizmann  
 Institute of Science, Rehovot 76100, Israel.  
 SO Genetics, (1998 Jul) Vol. 149, No. 3, pp. 1173-81.  
 Journal code: 0374636. ISSN: 0016-6731.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199808  
 ED Entered STN: 3 Sep 1998  
 Last Updated on STN: 29 Jan 1999

Entered Medline: 24 Aug 1998

AB A new mutagenesis assay system based on the phage 434 cI gene carried on a low-copy number plasmid was used to investigate the effect of UV light on intermolecular transposition of IS10. Inactivation of the target gene by IS10 insertion was detected by the expression of the tet gene from the phage 434 PR promoter, followed by Southern blot analysis of plasmids isolated from TetR colonies. UV irradiation of cells harboring the target plasmid and a donor plasmid carrying an IS10 element led to an increase of up to 28-fold in IS10 transposition. Each UV-induced transposition of IS10 was accompanied by fusion of the donor and acceptor plasmid into a cointegrate structure, due to coupled homologous recombination at the insertion site, similar to the situation in spontaneous IS10 transposition. UV radiation also induced transposition of IS10 from the chromosome to the target plasmid, leading almost exclusively to the integration of the target plasmid into the chromosome. UV induction of IS10 transposition did not depend on the umuC and uvrA gene product, but it was not observed in *lexA3* and *DeltarecA* strains, indicating that the SOS stress response is involved in regulating UV-induced transposition. IS10 transposition, known to increase the fitness of *Escherichia coli*, may have been recruited under the SOS response to assist in increasing cell survival under hostile environmental conditions. To our knowledge, this is the first report on the induction of transposition by a DNA-damaging agent and the SOS stress response in bacteria.

AB . . . low-copy number plasmid was used to investigate the effect of UV light on intermolecular transposition of IS10. Inactivation of the target gene by IS10 insertion was detected by the expression of the tet gene from the phage 434 PR promoter, followed by Southern blot analysis of plasmids. . . accompanied by fusion of the donor and acceptor plasmid into a cointegrate structure, due to coupled homologous recombination at the insertion site, similar to the situation in spontaneous IS10 transposition. UV radiation also induced transposition of IS10 from the chromosome to the target plasmid, leading almost exclusively to the integration. . .

L8 ANSWER 6 OF 11 MEDLINE on STN DUPLICATE 3

AN 1998305691 MEDLINE

DN PubMed ID: 9643538

TI Target specificity of insertion element IS30.

AU Olasz F; Kiss J; Konig P; Buzas Z; Stalder R; Arber W

CS Biozentrum der Universitat Basel, Abteilung Mikrobiologie, Basle, Switzerland.. olasz@hubi.abc.hu

SO Molecular microbiology, (1998 May) Vol. 28, No. 4, pp. 691-704.  
Journal code: 8712028. ISSN: 0950-382X.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 199809

ED Entered STN: 17 Sep 1998

Last Updated on STN: 29 Jan 1999

Entered Medline: 10 Sep 1998

AB The *Escherichia coli* resident mobile element IS30 has pronounced target specificity. Upon transposition, the element frequently inserts exactly into the same position of a preferred target sequence. Insertion sites in phages, plasmids and in the genome of *E. coli* are characterized by an exceptionally long palindromic consensus sequence that provides strong specificity for IS30 insertions, despite a relatively high level of degeneracy. This 24-bp-long region alone determines the attractiveness of the target DNA and the exact position of IS30 insertion. The divergence of a target site from the consensus and the occurrence of 'non-permitted' bases in



certain positions influence the target activity. Differences in attractiveness are emphasized if two targets are present in the same replicon, as was demonstrated by quantitative analysis. In a system of competitive targets, the oligonucleotide sequence representing the consensus of genomic IS30 insertion sites proved to be the most efficient target. Having compared the known insertion sites, we suppose that IS30-like target specificity, which may represent an alternative strategy in target selection among mobile elements, is characteristic of the insertion sequences IS3, IS6 and IS21, too.

AB The *Escherichia coli* resident mobile element IS30 has pronounced target specificity. Upon transposition, the element frequently inserts exactly into the same position of a preferred target sequence. Insertion sites in phages, plasmids and in the genome of . . . consensus sequence that provides strong specificity for IS30 insertions, despite a relatively high level of degeneracy. This 24-bp-long region alone determines the attractiveness of the target DNA and the exact position of IS30 insertion. The divergence of a target site from the consensus and the occurrence of 'non-permitted' bases in certain positions influence the. . .

L8 ANSWER 7 OF 11 MEDLINE on STN DUPLICATE 4  
 AN 94311866 MEDLINE  
 DN PubMed ID: 7518672  
 TI Retrotransposition of the *Drosophila* LINE I element can induce deletion in the target DNA: a simple model also accounting for the variability of the normally observed target site duplications.  
 AU Jensen S; Gassama M P; Heidmann T  
 CS Institut Gustave Roussy, CNRS URA147, Villejuif, France.  
 SO Biochemical and biophysical research communications, (1994 Jul 15) Vol. 202, No. 1, pp. 111-9.  
 Journal code: 0372516. ISSN: 0006-291X.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199408  
 ED Entered STN: 25 Aug 1994  
 Last Updated on STN: 29 Jan 1999  
 Entered Medline: 15 Aug 1994  
 AB Retrotransposition of the *Drosophila melanogaster* LINE I element normally generates target site duplications of variable length, as classically observed for most LINE elements. Using an I element "marked" with an indicator gene for in vivo detection of transposition that we previously developed, we show that deletion in the target DNA can also take place, as a direct consequence of I element transposition. We propose a simple model accounting for the generation of both target site duplications of variable length and target DNA deletions, which relies upon template switching of the LINE-encoded reverse transcriptase between single-strand DNA at the target site and the LINE template.  
 AB . . . length, as classically observed for most LINE elements. Using an I element "marked" with an indicator gene for in vivo detection of transposition that we previously developed, we show that deletion in the target DNA can also take place, as a direct consequence of I element transposition. We propose a simple model accounting for. . .

L8 ANSWER 8 OF 11 MEDLINE on STN DUPLICATE 5  
 AN 93211299 MEDLINE  
 DN PubMed ID: 8096321  
 TI Identification and characterization of IS1138, a transposable element from *Mycoplasma pulmonis* that belongs to the IS3 family.

AU Bhugra B; Dybvig K  
 CS Department of Microbiology, University of Alabama, Birmingham 35294.  
 NC AI31144 (NIAID)  
 P30 AI27767 (NIAID)  
 SO Molecular microbiology, (1993 Feb) Vol. 7, No. 4, pp. 577-84.  
 Journal code: 8712028. ISSN: 0950-382X.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 LA English  
 FS Priority Journals  
 OS GENBANK-Z16416  
 EM 199304  
 ED Entered STN: 14 May 1993  
 Last Updated on STN: 29 Jan 1999  
 Entered Medline: 27 Apr 1993  
 AB Insertion sequence (IS) elements are mobile genetic elements found in prokaryotes. We have identified a repetitive element from *Mycoplasma pulmonis*, a murine pathogen, that is similar to eubacterial IS elements. By subcloning a single strain of *M. pulmonis*, we isolated a variant clone in which the IS element had undergone an apparent transposition event. The nucleotide sequences of the element, designated IS1138, and the target site into which it inserted were determined. IS1138 consists of 1288 bp with 18 bp perfect terminal inverted repeats. Sequence analysis of the target site before and after insertion of IS1138 identified a 3 bp duplication of target DNA flanking the element. The predicted amino acids encoded by the major open reading frame of IS1138 share significant similarity with the transposases of the IS3 family. Southern hybridization analysis indicates that repetitive sequences similar to IS1138 are present in most, if not all, strains of *M. pulmonis*, but IS1138-like sequences were not detected in other mycoplasmal species.  
 AB . . . murine pathogen, that is similar to eubacterial IS elements. By subcloning a single strain of *M. pulmonis*, we isolated a variant clone in which the IS element had undergone an apparent transposition event. The nucleotide sequences of the element, designated IS1138, and the target site into which it inserted were determined. IS1138 consists of 1288 bp with 18 bp perfect terminal inverted repeats. Sequence analysis of the target site before and. . .  
 L8 ANSWER 9 OF 11 MEDLINE on STN  
 AN 84169549 MEDLINE  
 DN PubMed ID: 6324122  
 TI Characterization of insertions affecting the expression of the bacterio-opsin gene in *Halobacterium halobium*.  
 AU Pfeifer F; Friedman J; Boyer H W; Betlach M  
 NC GM31785-01 (NIGMS)  
 SO Nucleic acids research, (1984 Mar 12) Vol. 12, No. 5, pp. 2489-97.  
 Journal code: 0411011. ISSN: 0305-1048.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 LA English  
 FS Priority Journals  
 EM 198405  
 ED Entered STN: 19 Mar 1990  
 Last Updated on STN: 29 Jan 1999  
 Entered Medline: 2 May 1984  
 AB We have determined the sequence of the inverted repeats and duplicated target DNA of the halobacterial insertion elements ISH2 (520 bp), ISH23 (900 bp) and ISH24 (3000 bp) associated with

bacterio-opsin (bop) mutants. ISH2 has a perfect 19 bp inverted repeat (3,5), while both ISH23 and ISH24 have imperfect inverted repeats of 29 bp and 14 bp respectively. ISH23 was shown to be highly homologous to ISH50 (6). Variable lengths of duplicated target DNA are found when ISH2 and ISH23 (ISH50) transpose into different sites. A 550 bp DNA insert ("ISH25") reverts the Bop mutation caused by ISH24. "ISH25" lacks typical structural features of a transposable element. "ISH25" and ISH24 are found adjacent to each other upstream of the bop gene. An identical arrangement of "ISH25" and ISH24 is found in the cccDNA of *H. halobium* NRC817. Comparative sequence analysis of both areas suggests that the translocation of "ISH25" to the bop gene region occurred by a recombination event.

AB We have determined the sequence of the inverted repeats and duplicated target DNA of the halobacterial insertion elements ISH2 (520 bp), ISH23 (900 bp) and ISH24 (3000 bp) associated with bacterio-opsin (bop) mutants. ISH2 has a perfect. . . are found when ISH2 and ISH23 (ISH50) transpose into different sites. A 550 bp DNA insert ("ISH25") reverts the Bop mutation caused by ISH24. "ISH25" lacks typical structural features of a transposable element. "ISH25" and ISH24 are found adjacent to each other upstream of the bop gene. An identical arrangement of "ISH25". . .

L8 ANSWER 10 OF 11 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1985:223404 BIOSIS

DN PREV198579003400; BA79:3400

TI INSERTIONS OF TRANSPOSABLE ELEMENTS IN THE PROMOTER PROXIMAL REGION OF THE GENE CLUSTER FOR ESCHERICHIA-COLI PROTON-TRANSLOCATING ATPASE 8 BASE PAIR REPEAT GENERATED BY INSERTION OF IS-1.

AU KANAZAWA H [Reprint author]; KIYASU T; NOUMI T; FUTAI M; YAMAGUCHI K

CS DEPARTMENT OF MICROBIOLOGY, FACULTY OF PHARMACEUTICAL SCIENCES, OKAYAMA UNIVERSITY, OKAYAMA 700, JAPAN

SO Molecular and General Genetics, (1984) Vol. 194, No. 1-2, pp. 179-187. CODEN: MGGEAE. ISSN: 0026-8925.

DT Article

FS BA

LA ENGLISH

AB A plasmid pKY159 (Yamaguchi and Yamaguchi 1983) carrying a promoter proximal portion of the gene cluster of the H<sup>+</sup>-ATPase of *E. coli* causes growth inhibition of wild-type cells. Insertion of a transposable element in this plasmid released this inhibitory effect. In analyzing this inhibitory effect, the insertion points at the nucleotide-sequence level of transposable elements on 30 independent derivatives of pKY159 were determined. Insertions of IS1, IS5 and  $\gamma\delta$  were found between the promoter and the gene for a possible component of 14,000 daltons of the H<sup>+</sup>-ATPase. Of 31 insertions, 26 were of IS1 and were located at the same site, indicating that this site is a hotspot for IS1 insertion and that IS1 insertion is much more frequent than that of IS5 or  $\gamma\delta$  in this region. Four different sites for IS1 insertion were found; in 2 of these an 8 base pair (bp) duplicate of the target sequence (AAAAACGT and AAACGTTG) was generated, while in the other 2, a 9 bp duplicate was found. In all cases in this study the nucleotide sequence of IS1 was the same as that of IS1-K. In the 2 cases with an 8 bp duplicate in different sites, a common 6 bp sequence (AAACGT) was found. Apparently, generation of the 8 bp duplicate is related to the common sequence rather than a mutation in IS1 suggested by Iida et al. The essential length of the duplicate may be < 8 bp. A 6 bp sequence (GTGATG) homologous to the end portion of IS1 was found at the hotspot, but not at other sites, suggesting that this homology contributed to the high frequency of IS1 insertion. The direction of IS1 insertion at the hotspot was the same in 25 of 26 instances, suggesting that the direction of IS1 insertion is determined by the structure of the target and/or its

nearby sequence.

- AB. . . a promoter proximal portion of the gene cluster of the H<sup>+</sup>-ATPase of E. coli causes growth inhibition of wild-type cells. Insertion of a transposable element in this plasmid released this inhibitory effect. In analyzing this inhibitory effect, the insertion points at the nucleotide-sequence level of transposable elements on 30 independent derivatives of pKY159 were determined. Insertions of IS1, IS5 and  $\gamma\delta$  were found between the promoter. . . of IS1 insertion at the hotspot was the same in 25 of 26 instances, suggesting that the direction of IS1 insertion is determined by the structure of the target and/or its nearby sequence.

L8 ANSWER 11 OF 11 MEDLINE on STN DUPLICATE 6

AN 82162714 MEDLINE

DN PubMed ID: 6279310

TI A symmetrical six-base-pair target site sequence determines Tn10 insertion specificity.

AU Halling S M; Kleckner N

SO Cell, (1982 Jan) Vol. 28, No. 1, pp. 155-63.

Journal code: 0413066. ISSN: 0092-8674.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English

FS Priority Journals

EM 198206

ED Entered STN: 17 Mar 1990

Last Updated on STN: 29 Jan 1999

Entered Medline: 14 Jun 1982

- AB Transposon Tn10 inserts at many sites in the bacterial chromosome, but preferentially inserts at particular hotspots. We believe we have identified the target DNA signal responsible for this specificity. We have determined the DNA sequences of 11 Tn10 insertion sites and identified a particular 6 base pair (bp) symmetrical consensus sequence (GCTNAGC) common to those sites. The sequences at some sites differ from the consensus sequence but only in limited and well defined ways. The sequences at some sites differ from the consensus sequence than do sequences at other sites, and the consensus sequence and closely related sequences are generally absent from potential target regions where Tn10 is known not to insert. Other aspects of the target DNA can significantly influence the efficiency with which a particular target site sequence is used. The 6 bp consensus sequence is symmetrically located within the 9 bp target DNA sequence that is cleaved and duplicated during Tn10 insertion. This juxtaposition of recognition and cleavage sites plus the symmetry of the perfect consensus sequence suggest that the target DNA may be both recognized and cleaved by the symmetrically disposed subunits of a single protein, as suggested for type II restriction endonucleases. There is plausible homology between the consensus sequence and the very ends of Tn10, compatible with recognition of transposon ends and target DNA by the same protein. The sequences of actual insertion sites deviate from the perfect consensus sequence in a way which suggests that the 6 bp specificity determinant may be recognized through protein-DNA contacts along the major groove of the DNA double helix.

TI A symmetrical six-base-pair target site sequence determines Tn10 insertion specificity.

- AB Transposon Tn10 inserts at many sites in the bacterial chromosome, but preferentially inserts at particular hotspots. We believe we have identified the target. . .

=>

ANSWER 1 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:939428 CAPLUS

DN 145:329406

TI Detection of nucleic acid mismatch  
using Mu transposition and its use for identification  
of mutation and SNP in RNA

IN Yanagihara, Katsuhiko; Nakajima, Reiko

PA Kyoto University, Japan

SO Jpn. Kokai Tokkyo Koho, 22pp.

CODEN: JKXXAF

DT Patent

LA Japanese

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 2006238782	A	20060914	JP 2005-58584	20050303
PRAI	JP 2005-58584		20050303		

AB Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This invention provides a method of detection of nucleic acid mismatch using Mu terminal nucleic acid and Mu DNA transposases. The method comprises incubating DNA:RNA hybrid with Mu terminal sequence and Mu phage transposase, detection of transposition of Mu terminal sequence in DNA:RNA hybrid. The method provided in this invention can be used for reducing time consuming for detection mutation and SNP in RNA.

TI Detection of nucleic acid mismatch  
using Mu transposition and its use for identification  
of mutation and SNP in RNA

AB Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This invention provides a method of detection of nucleic acid mismatch using Mu terminal nucleic acid and Mu DNA transposases. The method comprises incubating DNA:RNA hybrid with Mu terminal sequence and Mu phage transposase, detection of transposition of Mu terminal sequence in DNA:RNA hybrid. The method provided in this invention can be used for reducing time consuming for detection mutation and SNP in RNA.

ST nucleic acid mismatch Mu transposition  
mutation SNP RNA; DNA RNA hybrid Mu terminal sequence  
transposase

IT DNA

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(RNA-hybrid; detection of nucleic acid  
mismatch using Mu transposition and its use  
for identification of mutation and SNP in genes)

IT RNA

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(SNP in; detection of nucleic acid  
mismatch using Mu transposition and its use  
for identification of mutation and SNP in genes)

IT Enterobacteria phage Mu

Genetic methods

Nucleic acid amplification (method)

(detection of nucleic acid  
mismatch using Mu transposition and its use  
for identification of mutation and SNP in genes)

IT Mutation

(detection of; detection of nucleic  
acid mismatch using Mu  
transposition and its use for identification of  
mutation and SNP in genes)

IT Nucleic acid bases

STN much

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(pairing, mismatch; detection of nucleic  
acid mismatch using Mu  
transposition and its use for identification of  
mutation and SNP in genes)

IT Genetic polymorphism  
(single nucleotide, detection of; detection of  
nucleic acid mismatch using Mu  
transposition and its use for identification of  
mutation and SNP in genes)

IT Nucleic acids  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(terminal, of Mu, labeled; detection of  
nucleic acid mismatch using Mu  
transposition and its use for identification of  
mutation and SNP in genes)

IT Enzymes, biological studies  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(transposases, of Mu pha; detection of  
nucleic acid mismatch using Mu  
transposition and its use for identification of  
mutation and SNP in genes)

IT 909611-37-6 909611-38-7 909611-39-8 909611-40-1 909611-41-2  
909611-42-3 909611-43-4 909611-44-5 909611-45-6 909611-46-7  
909611-47-8 909611-48-9 909611-49-0 909611-50-3 909611-51-4  
909611-52-5 909611-53-6 909611-54-7

RL: PRP (Properties)  
(unclaimed nucleotide sequence; detection of nucleic  
acid mismatch using Mu  
transposition and its use for identification of  
mutation and SNP in RNA)

L3 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2004:803873 CAPLUS

DN 141:290033

TI Methods for detecting genetic polymorphisms associated with cancer and for  
typing pathogenic microorganisms using mismatch-targeted Mu transposition  
for use in diagnosis

IN Yanagihara, Katsuhiko; Mizuuchi, Kiyoshi

PA United States Dept. of Health and Human Services, USA

SO U.S. Pat. Appl. Publ., 24 pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004191821	A1	20040930	US 2004-809688	20040326
PRAI	US 2003-457934P	P	20030328		

AB Phage Mu DNA transposes to duplex target DNA  
sites with limited sequence specificity. The present invention provides  
methods for detecting genetic polymorphisms associated with cancer and for  
typing pathogenic microbes using mismatch-targeted Mu transposition for  
use in diagnosis. Mu transposition exhibits a strong target site  
preference for all single-nucleotide mismatches. This finding has  
implications for the mechanism of transposition and provides a powerful  
tool for genomic research. A single mismatch could be  
detected as a preferred target of Mu transposition in  
the presence of 300,000-fold excess of non-mismatched sites.  
Both heterozygous and homozygous mutations in the cystic fibrosis  
transmembrane conductance regulator gene and single nucleotide  
polymorphism in HLA region were detected by Mu  
transposition mismatch anal. procedure.

AB Phage Mu DNA transposes to duplex target DNA sites with limited sequence specificity. The present invention provides methods for detecting genetic polymorphisms associated with cancer and for typing pathogenic microbes using mismatch-targeted Mu transposition for use in diagnosis. Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This finding has implications for the mechanism of transposition and provides a powerful tool for genomic research. A single mismatch could be detected as a preferred target of Mu transposition in the presence of 300,000-fold excess of non-mismatched sites. Both heterozygous and homozygous mutations in the cystic fibrosis transmembrane conductance regulator gene and single nucleotide polymorphism in HLA region were detected by Mu transposition mismatch anal. procedure.

ST mismatch targeted transposition phage Mu  
detection polymorphism mutation; gene HLA CFTR phage Mu  
transposition cancer diagnosis pathogen

L3 ANSWER 3 OF 8 MEDLINE on STN DUPLICATE 1  
AN 2003392175 MEDLINE  
DN PubMed ID: 12791691  
TI Effect of mutations in the C-terminal domain of Mu B on DNA binding and interactions with Mu A transposase.  
AU Coros Colin J; Sekino Yukiko; Baker Tania A; Chaconas George  
CS Department of Biochemistry, University of Western Ontario, London, Ontario N6A 5C1, Canada.  
NC GM49224 (NIGMS)  
SO The Journal of biological chemistry, (2003 Aug 15) Vol. 278, No. 33, pp. 31210-7. Electronic Publication: 2003-06-05.  
Journal code: 2985121R. ISSN: 0021-9258.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LA English  
FS Priority Journals  
EM 200311  
ED Entered STN: 22 Aug 2003  
Last Updated on STN: 11 Nov 2003  
Entered Medline: 10 Nov 2003

AB Bacteriophage Mu transposition requires two phage-encoded proteins, the transposase, Mu A, and an accessory protein, Mu B. Mu B is an ATP-dependent DNA-binding protein that is required for target capture and target immunity and is an allosteric activator of transpososome function. The recent NMR structure of the C-terminal domain of Mu B (Mu B223-312) revealed that there is a patch of positively charged residues on the solvent-exposed surface. This patch may be responsible for the nonspecific DNA binding activity displayed by the purified Mu B223-312 peptide. We show that mutations of three lysine residues within this patch completely abolish nonspecific DNA binding of the C-terminal peptide (Mu B223-312). To determine how this DNA binding activity affects transposition we mutated these lysine residues in the full-length protein. The full-length protein carrying all three mutations was deficient in both strand transfer and allosteric activation of transpososome function but retained ATPase activity. Peptide binding studies also revealed that this patch of basic residues within the C-terminal domain of Mu B is within a region of the protein that interacts directly with Mu A. Thus, we conclude that this protein segment contributes to both DNA binding and protein-protein contacts with the Mu transposase.

TI Effect of mutations in the C-terminal domain of Mu B on DNA binding and interactions with Mu A transposase.

AB Bacteriophage Mu transposition requires two phage-encoded proteins, the

transposase, Mu A, and an accessory protein, Mu B. Mu B is an ATP-dependent DNA-binding protein that is required for target capture and target immunity and is an allosteric activator of transpososome function. The recent. . . of three lysine residues within this patch completely abolish nonspecific DNA binding of the C-terminal peptide (Mu B223- 312). To determine how this DNA binding activity affects transposition we mutated these lysine residues in the full-length protein. The full-length protein carrying all three mutations was deficient in both strand transfer. . .

L3 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

AN 2002:670582 CAPLUS

DN 137:380570

TI Mismatch-targeted transposition of Mu: A new strategy to map genetic polymorphism

AU Yanagihara, Katsuhiko; Mizuuchi, Kiyoshi

CS. Laboratory of Molecular Biology, National Institute of Diabetes, Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD, 20892, USA

SO Proceedings of the National Academy of Sciences of the United States of America (2002), 99(17), 11317-11321

CODEN: PNASA6; ISSN: 0027-8424

PB National Academy of Sciences

DT Journal

LA English

AB Phage Mu DNA transposes to duplex target DNA

sites with limited sequence specificity. Here the authors demonstrate that Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This finding has implications for the mechanism of transposition and provides a powerful tool for genomic research. A single mismatch could be detected as a preferred target of Mu transposition in the presence of 300,000-fold excess of non-mismatched sites. The authors show the detection of both heterozygous and homozygous mutations in the cystic fibrosis transmembrane conductance regulator gene and single nucleotide polymorphism in HLA region by Mu transposition mismatch anal. procedure.

RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Phage Mu DNA transposes to duplex target DNA

sites with limited sequence specificity. Here the authors demonstrate that Mu transposition exhibits a strong target site preference for all single-nucleotide mismatches. This finding has implications for the mechanism of transposition and provides a powerful tool for genomic research. A single mismatch could be detected as a preferred target of Mu transposition in the presence of 300,000-fold excess of non-mismatched sites. The authors show the detection of both heterozygous and homozygous mutations in the cystic fibrosis transmembrane conductance regulator gene and single nucleotide polymorphism in HLA region by Mu transposition mismatch anal. procedure.

ST mismatch targeted transposition phage Mu

detection polymorphism mutation; human gene CFTR HLA variation detection phage Mu transposition

IT Gene, animal

RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)

(CFTR, mutation in; mismatch-targeted transposition of Mu in detection of human)

IT Gene, animal

RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)

(HLA-DP, DP $\alpha$ ; mismatch-targeted transposition of Mu in detection of polymorphism in)

IT Human



(gene CFTR mutation and HLA-DR $\alpha$  polymorphism detection by mismatch-targeted transposition of phage Mu)

IT Diagnosis  
(genetic; mismatch-targeted transposition of Mu in detection of genetic polymorphisms and mutations)

IT Enterobacteria phage Mu  
Genetic mapping  
Mutation  
(mismatch-targeted transposition of Mu in detection of genetic polymorphisms and mutations)

IT Genetic polymorphism  
(single nucleotide; mismatch-targeted transposition of Mu in detection of genetic polymorphisms and mutations)

IT Recombination, genetic  
(transposition; mismatch-targeted transposition of Mu in detection of genetic polymorphisms and mutations)

L3 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN  
AN 2002:355485 CAPLUS  
DN 137:180337  
TI Amplification and detection of transposon insertion flanking sequences using fluorescent MuAFLP  
AU Edwards, D.; Coghill, J.; Batley, J.; Holdsworth, M.; Edwards, K. J.  
CS University of Bristol, Bristol, UK  
SO BioTechniques (2002), 32(5), 1090-1092, 1094, 1096-1097 *order*  
CODEN: BTNQDO; ISSN: 0736-6205  
PB Eaton Publishing Co.  
DT Journal  
LA English  
AB The amplification of transposon insertion flanking sequences is the basis of a variety of techniques used for the detection and characterization of specific transposon insertion events. In this study the authors report on the development of a method for the efficient size determination and quantification of amplified genomic sequences that flank Mutator (Mu) transposon insertions in maize. Using this detection method, the authors have been able to optimize Mu insertion site amplification and to assess amplification from increasingly complex templates representing increasing nos. of Mu-active maize plants. This detection method should be applicable for the characterization of transposon or transgene insertion events in a wide variety of organisms.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Amplification and detection of transposon insertion flanking sequences using fluorescent MuAFLP

AB The amplification of transposon insertion flanking sequences is the basis of a variety of techniques used for the detection and characterization of specific transposon insertion events. In this study the authors report on the development of a method for the efficient size determination and quantification of amplified genomic sequences that flank Mutator (Mu) transposon insertions in maize. Using this detection method, the authors have been able to optimize Mu insertion site amplification and to assess amplification from increasingly complex templates representing increasing nos. of Mu-active maize plants. This detection method should be applicable for the characterization of transposon or transgene insertion events in a wide variety of organisms.

IT Primers (nucleic acid)  
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(DNA, Mu and MseI adapter-specific primers; amplification and detection of transposon Mutator (Mu) insertion flanking sequences in maize plants using fluorescent MuAFLP technique)

IT Transposons  
 RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 (Mu element; amplification and detection of transposon Mutator (Mu) insertion flanking sequences using fluorescent MuAFLP, method involves DNA digestion, modified adapter ligation, magnetic bead selection and PCR-AFLP)

IT Zea mays  
 (amplification and detection of transposon Mutator (Mu) insertion flanking sequences in maize plants using fluorescent MuAFLP technique)

IT AFLP (amplified fragment length polymorphism)  
 PCR (polymerase chain reaction)  
 (amplification and detection of transposon Mutator (Mu) insertion flanking sequences using fluorescent MuAFLP, method involves DNA digestion, modified adapter ligation, magnetic bead selection and PCR-AFLP)

IT Genetic methods  
 (fluorescent MuAFLP; amplification and detection of transposon Mutator (Mu) insertion flanking sequences using fluorescent MuAFLP, method involves DNA digestion, modified adapter ligation, magnetic bead selection and PCR-AFLP)

IT Magnetic materials  
 (magnetic streptavidin beads; amplification and detection of transposon Mutator (Mu) insertion flanking sequences using fluorescent MuAFLP, method involves DNA digestion, modified adapter ligation, magnetic bead selection and PCR-AFLP)

IT Oligodeoxyribonucleotides  
 RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)  
 (modified adapter; amplification and detection of transposon Mutator (Mu) insertion flanking sequences using fluorescent MuAFLP, method involves DNA digestion, modified adapter ligation, magnetic bead selection and PCR-AFLP)

IT DNA sequences  
 (of maize Mu terminal sequences, and their use in design of PCR primers)

IT DNA  
 RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (primer, Mu and MseI adapter-specific primers; amplification and detection of transposon Mutator (Mu) insertion flanking sequences in maize plants using fluorescent MuAFLP technique)

IT DNA  
 Genetic element  
 RL: ANT (Analyte); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (sequences flanking transposon insertion sites; amplification and detection of transposon Mutator (Mu) insertion flanking sequences in maize plants using fluorescent MuAFLP technique)

IT Mutagenesis  
 (transposon; amplification and detection of transposon Mutator (Mu) insertion flanking sequences in maize plants using fluorescent MuAFLP technique, potential use of method for characterization of insertion events in mutagenesis systems)

L3 ANSWER 6 OF 8 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
AN 1990:172136 BIOSIS  
DN PREV199089089306; BA89:89306  
TI OCCURRENCE OF DNA SEQUENCES HOMOLOGOUS TO THE MAIZE MU  
TRANSPOSABLE ELEMENT IN WHEAT AND OTHER CEREAL SPECIES.  
AU SPARVOLI F [Reprint author]; PATROSSO M C; VIOTTI A; POGNA N E  
CS IST SPERIMENTALE CEREALICOLTURA, VIA MOLINO 3, 20079 S ANGELO LODIGIANO,  
ITALY  
SO Journal of Genetics and Breeding, (1989) Vol. 43, No. 4, pp. 237-244.  
ISSN: 0394-9257.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 10 Apr 1990

Last Updated on STN: 10 Apr 1990

AB DNA sequences related to Mu, a family of  
transposable elements isolated from "Mutator" maize  
stocks, have been detected in Triticum species as well as in  
several cereals representative of the different tribes of gramineae.  
Southern blot analysis of EcoRI or TaqI digested DNAs of wheat species  
showed that the two component parts of Mu element, i.e. the internal  
region and the long terminal inverted repeats, are always in association  
with one another. Moreover, no fragment restriction polymorphism has been  
found among diploid or polyploid wheats as well as among several common  
wheat cultivars restricted with BclI, KpnI and EcoRI. The results suggest  
that Mu-like elements are old components of wheat genomes and have lost  
their transposition activity, if any, prior to the divergence of the  
ancestors of the A, B and D genomes. The A-, B- and D-genome diploid  
wheats each contains about 4-6 Mu-like elements, the tetraploid and  
hexaploid species having twice and three times more elements,  
respectively. A higher copy number of Mu-like elements has been found in  
rye and triticale.

TI OCCURRENCE OF DNA SEQUENCES HOMOLOGOUS TO THE MAIZE MU  
TRANSPOSABLE ELEMENT IN WHEAT AND OTHER CEREAL SPECIES.

AB DNA sequences related to Mu, a family of  
transposable elements isolated from "Mutator" maize  
stocks, have been detected in Triticum species as well as in  
several cereals representative of the different tribes of gramineae.  
Southern blot analysis of. . .

L3 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

AN 1984:80685 CAPLUS

DN 100:80685

TI Use of Mu phages to isolate transposon insertions juxtaposed to given  
genes of Escherichia coli

AU Cronan, John E., Jr.

CS Dep. Microbiol., Univ. Illinois, Urbana, IL, 61801, USA

SO Current Microbiology (1983), 9(5), 245-51

CODEN: CUMIDD; ISSN: 0343-8651

DT Journal

LA English

AB The small sizes of the DNA fragments transduced by lysates of phage Mu and  
of mixed lysates of Mu and mini-Mu18A-1 (an internally deleted Mu phage)  
provide a method for the selection of insertions of transposon Tn10  
located very close to given E. coli genes. Generalized transduction with  
Mu lysates selected for those insertions located within 38 kilobase pairs  
of the gene of interest, whereas insertions located within .apprx.1/2 that  
distance are directly selected by use of mini-Mu phages. Use of these  
transduction systems avoids screening of individual colonies by phage P1  
transduction for those transposon insertions closely linked to a given  
gene. Such insertions are useful for localized mutagenesis and for in  
vitro mol. cloning.

IT Mutation

(insertion, from transposon Tn10, in Escherichia coli, phage Mu in detection of)

IT Gene and Genetic element, microbial  
 RL: BIOL (Biological study)  
 (transposon Tn10, insertion of, in Escherichia coli DNA,  
 phage Mu in separation of)

L3 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3  
 AN 1981:135937 CAPLUS  
 DN 94:135937  
 TI In vitro insertions and deletions in the G segment of phage Mu  
 DNA do not abolish the inversion process  
 AU Clayton, R.; Schumann, W.; Bade, E. G.  
 CS Fak. Biol., Univ. Konstanz, Konstanz, D-7750, Fed. Rep. Ger.  
 SO Virology (1981), 109(2), 267-80  
 CODEN: VIRLAX; ISSN: 0042-6822  
 DT Journal  
 LA English  
 AB The hybrid plasmid pKN56, which contains the right-end PstI·B  
 fragment of phage Mu DNA with its invertible G  
 segment, was used to test if the phys. integrity of the G segment is  
 required for the inversion process. Insertion of a 4.1-megadalton (Md)  
 fragment encoding resistance to kanamycin into the KpnI site located in  
 the G segment did not abolish the invertibility of the G segment in the  
 resulting plasmids (pKN72, pKN73). Insertion of addition non-Mu  
 DNA up to a total mol. weight of .apprx.10 + 106 also did not  
 impair G inversion. In vitro shortening of the G segment to .apprx.1/2  
 its size by removal of the internal HpaI fragment also failed to alter the  
 inversion process. Inversion also occurred with normal frequency in  
 Escherichia coli mutants hip, himA, and himB, which affect integration of  
 phage λ and Mu development,. By measuring the curing efficiency of  
 the enlarged and shortened plasmids in a polA<sup>ts</sup> strain grown at  
 43°, it was shown that no transposition of the G segment occurs at  
 levels >10<sup>-5</sup> for pKN72 and pKN73 and >10<sup>-7</sup> for pKN119. However,  
 transposition of Tn601 inserted in the G segment could  
 not be detected. The KpmI and HpaI restriction sites in the G  
 segment were partially mapped by marker rescue expts. with amber mutants  
 in the Mu genes S and U. Genes S and U were expressed from different  
 promoters, as shown by complementation.

TI In vitro insertions and deletions in the G segment of phage Mu  
 DNA do not abolish the inversion process

AB The hybrid plasmid pKN56, which contains the right-end PstI·B  
 fragment of phage Mu DNA with its invertible G  
 segment, was used to test if the phys. integrity of the G segment is  
 required for the inversion process. Insertion of a 4.1-megadalton (Md)  
 fragment encoding resistance to kanamycin into the KpnI site located in  
 the G segment did not abolish the invertibility of the G segment in the  
 resulting plasmids (pKN72, pKN73). Insertion of addition non-Mu  
 DNA up to a total mol. weight of .apprx.10 + 106 also did not  
 impair G inversion. In vitro shortening of the G segment to .apprx.1/2  
 its size by removal of the internal HpaI fragment also failed to alter the  
 inversion process. Inversion also occurred with normal frequency in  
 Escherichia coli mutants hip, himA, and himB, which affect integration of  
 phage λ and Mu development,. By measuring the curing efficiency of  
 the enlarged and shortened plasmids in a polA<sup>ts</sup> strain grown at  
 43°, it was shown that no transposition of the G segment occurs at  
 levels >10<sup>-5</sup> for pKN72 and pKN73 and >10<sup>-7</sup> for pKN119. However,  
 transposition of Tn601 inserted in the G segment could  
 not be detected. The KpmI and HpaI restriction sites in the G  
 segment were partially mapped by marker rescue expts. with amber mutants  
 in the Mu genes S and U. Genes S and U were expressed from different  
 promoters, as shown by complementation.

ST phage Mu DNA G segment inversion

IT Virus, bacterial  
(mu, DNA of, insertions and deletions in G segment  
of, inversion in relation to)

=>